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EXAMINER	
DUNSTON, JENNIFER ANN	
ART UNIT	**PAPER NUMBER**
1636	

SHORTENED STATUTORY PERIOD OF RESPONSE	MAIL DATE	DELIVERY MODE
3 MONTHS	02/27/2007	PAPER

Please find below and/or attached an Office communication concerning this application or proceeding.

If NO period for reply is specified above, the maximum statutory period will apply and will expire 6 MONTHS from the mailing date of this communication.

T4

Office Action Summary	Application No.	Applicant(s)	
	10/663,002	MUKHERJEE ET AL.	
	Examiner	Art Unit	
	Jennifer Dunston	1636	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *19 December 2006*.

2a)☒ This action is **FINAL**. 2b)☐ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1-5 and 17-24* is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *1-3,5,17-19 and 21-24* is/are rejected.

7)☒ Claim(s) *4 and 20* is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☒ The drawing(s) filed on *16 September 2003* is/are: a)☒ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☐ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date _____.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____.
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

DETAILED ACTION

This action is in response to the amendment, filed 12/19/2006, in which claims 6-16 were canceled, claims 1, 4 and 5 were amended, and claims 17-24 were newly added. Currently claims 1-5 and 17-24 are pending.

Applicant's arguments have been thoroughly reviewed, but are not persuasive for the reasons that follow. Any rejections and objections not reiterated in this action have been withdrawn. **This action is FINAL.**

Election/Restrictions

Applicant elected Group I without traverse in the reply filed on 6/29/2006. Claims 1-5 and 17-24 read on elected Group I and are currently under consideration.

Claim Objections – Double Patenting (Warning)

Applicant is advised that should claim 2 be found allowable, claim18 will be objected to under 37 CFR 1.75 as being a substantial duplicate thereof. When two claims in an application are duplicates or else are so close in content that they both cover the same thing, despite a slight difference in wording, it is proper after allowing one claim to object to the other as being a substantial duplicate of the allowed claim. See MPEP § 706.03(k). Both claim 2 and claim 18 depend from claim 1. The claims are identical in scope. This is a new objection, necessitated by the addition of new claim 18 in the reply filed 12/19/2006.

Claim Rejections - 35 USC § 112

The following is a quotation of the first paragraph of 35 U.S.C. 112:

> The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

Claims 1-3, 5, 17-19 and 21-24 are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the written description requirement. The claim(s) contains subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention. This rejection was applied to claims 1-5 in the Office action mailed 9/19/2006 and has been altered to address the amendments to the claims in the reply filed 12/19/2006.

Claim 21 is drawn to the measurement of a "PPAR responsive gene" in any type of cell obtained from any species of organism. Claim 1 is drawn to the measurement of "a PPAR responsive gene selected from the group consisting of pyruvate dehydrogenase kinase-4 (PDK-4) and adipocyte differentiation relating protein (ADRP)." Claim 17 is drawn to the measurement of ADRP, where ADRP is a PPAR responsive gene. Claims 2, 18 and 22 further limit the isoform of PPAR to PPAR-α, PPAR-β(δ), or PPAR-γ, but do not limit the type of cell or organism. Claims 3, 19 and 23 limit the cell to a mammalian cell but do not specify the type of cell. Claim 24 further limits the mammalian cell to a human proximal tubule derived cell (HK-2). Claims 21, 23 and 24 do not limit the gene assayed or isoform of PPAR. Thus, the claims encompass a set of PPAR-α, responsive genes from any organism and any cell type, PPAR-β(δ)

responsive genes from any organism and any cell type, or PPAR-γ responsive genes from any organism and any cell type. Further, the claims encompass any PPAR responsive gene present in a human proximal tubule derived cell that is an HK-2 cell. Moreover, the claims encompass any PDK-4 or ADRP gene from any organism and cell type. Accordingly, the claims encompass a large genus of PPAR responsive genes.

To provide adequate written description and evidence of possession of a claimed genus, the specification must provide sufficient distinguishing identifying characteristics of the genus. The factors to be considered include disclosure of a complete or partial structure, physical and/or chemical properties, functional characteristics, structure/function correlation, and any combination thereof. The specification teaches that there are species differences and cell type differences in the expression of PPAR responsive genes (e.g. page 2, lines 15-32; page 3, lines 4-7; page 10, lines 20-24; paragraph bridging pages 11-12; Figure 1B). The specification describes PDK-4 and ADRP as PPAR responsive genes in human HK-2 cells (e.g. page 3, lines 23-26; Figures 1A and 2A). PDK-4 is further classified as a PPAR responsive gene in hamster kidney and hamster liver (e.g. page 3, lines 26-32; Figure 4). However, increased PDK-4 and ADRP expression was not consistent among all cell lines tested: HK-2, SW872, LNCaP, ACHN, HepG2 and Caki-1 (e.g. paragraph bridging pages 11-12; Figure 1B). Given the variability seen between species and cell types, the disclosure of PDK-4 as a PPAR responsive gene in human HK-2 cells and hamster kidney and liver cells and of ADRP as a PPAR responsive gene in human HK-2 cells, there is not a significant structure/function correlation that would allow one to use PDK-4 and/or ADRP in a representative number of species and cell types encompassed by the claims.

Even if one accepts that the examples described in the specification meet the claim limitations of the rejected claims with regard to structure and function, the examples are only representative of small set of genes that may be used in HK-2 cells or hamster liver or kidney. The results are not necessarily predictive of the ability to use these genes in other species or cell types. Thus, it is impossible for one to extrapolate from the few examples described herein those genes that would necessarily meet the structural/functional characteristics of the rejected claims.

The prior art does not appear to offset the deficiencies of the instant specification in that it does not describe a representative number of PPAR responsive genes for each of the PPAR isoforms, species and cell types encompassed by the claims. For example, Way et al (Endocrinology, Vol. 142, No. 3, pages 1269-1277, March 2001) teach genes that are up- or down-regulated in response to PPARγ activation in rat (e.g. Table 2). However, the genes are not consistently regulated across all cell types (e.g. Table 2). This lack of structure function correlation requires the identity of PPAR responsive genes to be experimentally determined for each condition: PPAR isoform (α, β(δ) and γ), organism (e.g. Drosophila, C. elegans, cow, pig, rat, mouse, human, etc.) and cell type (e.g. fibroblast, muscle, hepatocyte, kidney epithelial cell, neuron, etc.).

Vas-Cath Inc. v. Mahurkar, 19USPQ2d 1111, clearly states, "applicant must convey with reasonable clarity to those skilled in the art that, as of the filing date sought, he or she was in possession of *the invention*. The invention is, for purposes of the 'written description' inquiry, *whatever is now claimed.*" (See page 1117.) The specification does not "clearly allow persons of ordinary skill in the art to recognize that [he or she] invented what is now is claimed." (See *Vas-Cath* at page 1116). As discussed above, the skilled artisan cannot envision the detailed

chemical structure of the encompassed genus of PPAR responsive genes, and therefore conception is not achieved until reduction to practice has occurred, regardless of the complexity or simplicity of the method of isolation or identification. Adequate written description requires more than a mere statement that it is part of the invention and reference to a potential method of isolating it. The compound itself is required. See *Fiers v. Revel*, 25USPQ2d 1601 at 1606 (CAFC 1993) and *Amgen Inc. v. Chugai Pharmaceutical Co. Ltd.*, 18USPQ2d 1016.

One cannot describe what one has not conceived. See *Fiddes v. Baird*, 30 USPQ2d 1481 at 1483. In *Fiddes*, claims directed to mammalian FGF's were found to be unpatentable due to lack of written description for that broad class. The specification provided only the bovine sequence.

Given the very large genus of PPAR responsive genes encompassed by the rejected claims, and given the limited description provided by the prior art and specification with regard to the specific PPAR responsive genes for each isoform and a representative number of organisms and cell types, the skilled artisan would not have been able to envision a sufficient number of specific embodiments that meet the functional limitations of the claims to describe the broadly claimed genus of PPAR responsive genes. Thus, there is no structural/functional basis provided by the prior art or instant specification for one of skill in the art to envision those PPAR responsive genes that satisfy the functional limitations of the claims. Therefore, the skilled artisan would have reasonably concluded applicants were not in possession of the claimed invention for claims 1-3, 5, 17-19 and 21-24.

Response to Arguments - 35 USC § 112

The rejection of claims 1-5 under 35 U.S.C. 112, second paragraph, has been withdrawn in view of Applicant's amendment to the claims in the reply filed 12/19/2006.

With respect to the rejection of claims 1-3, 5, 17-19 and 21-24 are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the written description requirement, Applicant's arguments filed 12/19/2006 have been fully considered but they are not persuasive.

The response asserts that the Office has not made a *prima facie* case with regard to the instant rejection, because the instant claims are method claims. This is not found persuasive, because one must be in possession of a set of PPAR responsive genes and the cell type for which those genes are PPAR responsive genes in order to carry out the claimed method. One must know the mRNA sequences of those PPAR responsive genes such that one could perform the step of determining an mRNA transcript level. If the specification or prior art does not put one in possession of the set of genes and the mRNA sequences for a particular cell type, then the claimed invention lacks description.

The response asserts that Applicant provides two examples of PPAR responsive genes (PDK-4 and ADRP), and one could identify other genes for use in the assay. Further, the response points to the prior art of record, which teaches some PPAR responsive genes in some cell types of some species of organism. "A patentee will not be deemed to have invented species sufficient to constitute the genus by virtue of having disclosed a single species when ... the evidence indicates ordinary artisans could not predict the operability in the invention of any species other than the one disclosed." *In re Curtis*, 354 F.3d 1347, 1358, 69 USPQ2d 1274, 1282 (Fed. Cir. 2004). Given the fact that PDK-4 and ADRP are not PPAR responsive genes in every

cell type tested in the instant specification, one could not extrapolate those examples available in the prior art or instant specification to envision a representative number of species within the claimed genus with respect to PPAR genes in a particular cell type or the cell types for which PDK-4 and/or ADRP are PPAR responsive genes.

For these reasons, and the reasons made of record in the previous office actions, the rejection is maintained.

Claim Rejections - 35 USC § 102

The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

> A person shall be entitled to a patent unless –
>
> (a) the invention was known or used by others in this country, or patented or described in a printed publication in this or a foreign country, before the invention thereof by the applicant for a patent.
>
> (b) the invention was patented or described in a printed publication in this or a foreign country or in public use or on sale in this country, more than one year prior to the date of application for patent in the United States.

Claims 21-23 are rejected under 35 U.S.C. 102(b) as being anticipated by Way et al (Endocrinology, Vol. 142, No. 3, pages 1269-1277, March 2001; see the entire reference). This rejection was applied to claims 1-3 in the Office action mailed 9/19/2006 and has been altered to address the amendments to the claims in the reply filed 12/19/2006.

Regarding claim 21, Way et al teach a method comprising the steps of (i) treating ZDF rats for 7 days with either GW1929 or vehicle alone, (ii) determining the level of a plurality of mRNA transcripts in cells exposed to GW1929 or vehicle alone with GeneCalling mRNA profiling technology, and comparing the level of transcripts in cells exposed to GW1929 and cells exposed to vehicle alone (e.g. paragraph bridging pages 1270-1271). GW1929 is a

tyrosine-based PPARγ agonist (e.g. paragraph bridging pages 1270-1271). The genes that are upregulated or downregulated by GW1929 and are thus PPARγ responsive genes are disclosed in Table 2 (page 1272). The method of claim 21 comprises the step of contacting a cell with a single dose of a test compound. As written, the claim can include additional steps such as the method further comprising the addition of a second dose. Further, Way et al use a single dose of the GW1929 compound: 5.0 mg/kg (e.g. page 1270, Experimental animals and protocols).

Regarding claim 22, Way et al teach that GW1929 is a selective PPARγ modulator (e.g. paragraph bridging pages 1269-1270).

Regarding claim 23, Way et al teach the method in rat cells, which are mammalian cells.

Claims 21-23 are rejected under 35 U.S.C. 102(b) as being anticipated by Jiang et al (Journal of Lipid Research, Vol. 42, pages 716-724, May 2001; see the entire reference). This rejection was applied to claims 1-3 in the Office action mailed 9/19/2006 and has been altered to address the amendments to the claims in the reply filed 12/19/2006.

Regarding claim 21, Jiang et al teach a method comprising culturing SW cells in the presence of clofibrate for 0-72 h, isolating mRNA for $cPLA_2$ and COX-2, determining the level of $cPLA_2$ and COX-2 mRNA at various time intervals, including 0, 48 and 72 h, and comparing the amount of mRNA at each of the time intervals (e.g. page 720, Up-regulation of $cPLA_2$ and COX-2 mRNA by clofibrate; Figures 3 and 4). The cells tested between 0 and 48 h receive a single dose of clofibrate (e.g. page 717, paragraph bridging columns).

Regarding claim 22, Clofibrate is a known PPARα agonist (e.g. page 722, right column, 1st full paragraph).

Regarding claim 23, the SW cells used by Jiang et al are preadipocyte SW 872 (SW) cells, derived from human liposarcoma and are thus mammalian cells (e.g. page 717, Cell culture and radiolabeling).

Claims 21-23 are rejected under 35 U.S.C. 102(b) as being anticipated by Crabb et al (Alcoholism: Clinical and Experimental Research, Vol. 25, No. 7, pages 945-952, July 2001; see the entire reference). This rejection was applied to claims 1-3 in the Office action mailed 9/19/2006 and has been altered to address the amendments to the claims in the reply filed 12/19/2006.

Regarding claim 21, Crabb et al teach a method comprising treating rats with and without clofibrate, measuring the level of *ALDH2* mRNA expression in rats with and without treatment with clofibrate, and comparing the levels of ALDH2 mRNA expression (e.g. page 949, *Effect of Clofibrate Treatment on ALDH2 Expression in Rat Liver*). The method of claim 21 comprises the step of contacting a cell with a single dose of a test compound. As written, the claim can include additional steps such as the method further comprising the addition of a second dose. Further, Crabb et al use a single dose of the clofibrate compound: 0.25% w/w (e.g. page 946, Animals).

Regarding claim 22, Crabb et al teach the analysis of PPARα (e.g. page 949, *Effect of Clofibrate Treatment on ALDH2 Expression in Rat Liver*).

Regarding claim 23, the cells used in the method of Crabb et al are rat cells and are thus mammalian cells (e.g. page 949, *Effect of Clofibrate Treatment on ALDH2 Expression in Rat Liver*).

Claims 21-23 are rejected under 35 U.S.C. 102(b) as being anticipated by Wu et al (Diabetes, Vol. 48, pages 1593-1599, August 1999; see the entire reference). This rejection was applied to claims 1-3 in the Office action mailed 9/19/2006 and has been altered to address the amendments to the claims in the reply filed 12/19/2006.

Regarding claim 21, Wu et al teach a method comprising treating rats with or without WY-14,643 for 3 days, measuring the level of PDK4 mRNA from cells with and without WY-14,643 treatment, and comparing the levels of mRNA between treated and untreated cells to determine that WY-14,643 increases PDK4 mRNA expression (e.g. page 1596, left column, 1st full paragraph; Figure 5). The method of claim 21 comprises the step of contacting a cell with a single dose of a test compound. As written, the claim can include additional steps such as the method further comprising the addition of a second dose. Further, Wu et al use a single dose of the WY-14,643 compound: 0.1 % (e.g. page 946, Animals).

Regarding claim 22, Wu et al teach that WY-14,643 is a PPARα agonist (e.g. page 1598, left column, 2nd full paragraph).

Regarding claim 23, Wu et al teach the use of cells obtained from rat (i.e. a mammal) (e.g. page 1596, left column, 1st full paragraph; Figure 5).

Claims 1-3, 5, 17-19 and 21-23 are rejected under 35 U.S.C. 102(b) as being anticipated by Gupta et al (The Journal of Biological Chemistry, Vol. 276, No. 32, pages 29681-29687, August 2001; see the entire reference). This is a new rejection, necessitated by the amendment of claims 1 and 5, and the addition of claims 17-24 in the reply filed 12/19/2006.

Regarding claims 1, 5, 17 and 21, Gupta et al teach a method comprising treating the MOSER S (M-S) colon carcinoma cell line *in vitro* with a single dose of rosiglitazone, GW7845, GW9662, GW7647 or GW1514, measuring the level of ADRP (a.k.a., adipose differentiation-related protein, and acidophilin) mRNA in treated and untreated cells to determine that the compounds increase or decrease ADRP expression through PPAR proteins (e.g. page 29682, Materials and Cell Culture, Receptor Ligands, Oligonucleotide Microarray Screening, and Northern Hybridization Analysis; pages 29683-29684, Identification of PPARγ Target Genes Using Microarrays; page 29684, right column, last full paragraph; Figure 3).

Regarding claims 2, 18 and 22, Gupta et al teach that rosiglitazone is a PPARγ agonist, GW7845 is a PPARγ agonist, GW9662 is a PPARγ antagonist, GW7647 is a PPARα agonist, and GW1514 is a PPARδ agonist (e.g. page 29682, Receptor Ligands).

Regarding claims 3, 19 and 23, the MOSER S (M-S) colon carcinoma cell line used by Gupta et al is a mammalian cell line (e.g. page 29682, Materials and Cell Culture).

Response to Arguments - 35 USC § 102

The rejection of claims 1-3 under 35 U.S.C. 102(b) as being anticipated by Way et al has been withdrawn in view of Applicant's amendment to the claims in the reply filed 12/19/2006.

With respect to the application of the Way et al reference under 35 U.S.C. 102(b), Applicant's arguments filed 12/19/2006 have been fully considered with respect to the instantly rejected claims but they are not persuasive.

The response asserts that claims 21-24 limit the claims to a single dose of PPAR agonist, which Way et al do not teach. This is not found persuasive, because the claims, as written, can

include additional steps such as the method further comprising the addition of a second dose. Further, Way et al use a single dose of the GW1929 compound: 5.0 mg/kg (e.g. page 1270, Experimental animals and protocols).

The response acknowledges that Way et al teach an *in vivo* system, and do not teach an *in vitro* cell based assay. In response to applicant's argument that the references fail to show certain features of applicant's invention, it is noted that the features upon which applicant relies (i.e., *in vitro* cell based assay) are not recited in the rejected claim(s), claims 21-23. Although the claims are interpreted in light of the specification, limitations from the specification are not read into the claims. See *In re Van Geuns*, 988 F.2d 1181, 26 USPQ2d 1057 (Fed. Cir. 1993).

For these reasons, and the reasons made of record in the previous office actions, the rejection is maintained with respect to claims 21-23.

The rejection of claims 1-3 under 35 U.S.C. 102(b) as being anticipated by Jiang et al has been withdrawn in view of Applicant's amendment to the claims in the reply filed 12/19/2006.

With respect to the application of the Jiang et al reference under 35 U.S.C. 102(b), Applicant's arguments filed 12/19/2006 have been fully considered with respect to the instantly rejected claims but they are not persuasive.

The response asserts that claims 21-24 limit the claims to a single dose of PPAR agonist, which Jiang et al do not teach. This is not found persuasive, because the cells tested between 0 and 48 h receive a single dose of clofibrate (e.g. page 717, paragraph bridging columns).

For these reasons, and the reasons made of record in the previous office actions, the rejection is maintained with respect to claims 21-23.

The rejection of claims 1-3 under 35 U.S.C. 102(b) as being anticipated by Crabb et al has been withdrawn in view of Applicant's amendment to the claims in the reply filed 12/19/2006.

With respect to the application of the Crabb et al reference under 35 U.S.C. 102(b), Applicant's arguments filed 12/19/2006 have been fully considered with respect to the instantly rejected claims but they are not persuasive.

The response asserts that claims 21-24 limit the claims to a single dose of PPAR agonist, which Crabb et al do not teach. This is not found persuasive, because the claims, as written, can include additional steps such as the method further comprising the addition of a second dose. Further, Crabb et al use a single dose of the clofibrate compound: 0.25% w/w (e.g. page 946, Animals).

For these reasons, and the reasons made of record in the previous office actions, the rejection is maintained with respect to claims 21-23.

The rejection of claims 1-3 and 5 under 35 U.S.C. 102(b) as being anticipated by Wu et al has been withdrawn in view of Applicant's amendment to the claims in the reply filed 12/19/2006.

With respect to the application of the Wu et al reference under 35 U.S.C. 102(b), Applicant's arguments filed 12/19/2006 have been fully considered with respect to the instantly rejected claims but they are not persuasive.

The response asserts that claims 21-24 limit the claims to a single dose of PPAR agonist, which Wu et al do not teach. This is not found persuasive, because the claims, as written, can include additional steps such as the method further comprising the addition of a second dose.

Further, Wu et al use a single dose of the WY-14,643 compound: 0.1 % (e.g. page 946, Animals).

For these reasons, and the reasons made of record in the previous office actions, the rejection is maintained with respect to claims 21-23.

Conclusion

No claims are allowed.

Claims 4 and 20 are objected to as being dependent upon a rejected base claim.

Applicant's amendment necessitated the new ground(s) of rejection presented in this Office action. Accordingly, **THIS ACTION IS MADE FINAL**. See MPEP § 706.07(a). Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no event, however, will the statutory period for reply expire later than SIX MONTHS from the date of this final action.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Jennifer Dunston whose telephone number is 571-272-2916. The examiner can normally be reached on M-F, 9 am to 5 pm.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Remy Yucel can be reached on 571-272-0781. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free). If you would like assistance from a USPTO Customer Service Representative or access to the automated information system, call 800-786-9199 (IN USA OR CANADA) or 571-272-1000.

Jennifer Dunston, Ph.D.
Examiner
Art Unit 1636

jad

CELINE QIAN, PH.D.
PRIMARY EXAMINER